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RECOMMENDED IN THE 2025 KDIGO GUIDELINES[1]

KINPEYGO® 4 mg

Modified-release hard capsules budesonide

Clinical Summary

Updated NICE guidelines recommend disease-modifying Kinpeygo® as an option to treat primary IgA nephropathy in adults with UPCR ≥ 90 mg/mmol or protein excretion of ≥ 1.0 g/day[2]

Both NICE and KDIGO recommend treatment of IgA nephropathy with Kinpeygo®, alongside optimised standard care[1,2]

Kinpeygo® is indicated for the treatment of adults with primary IgA nephropathy with a urine protein excretion ≥ 1.0 g/day (or UPCR ≥ 0.8 g/g, equivalent to ≥ 90 mg/mmol)[2,3]

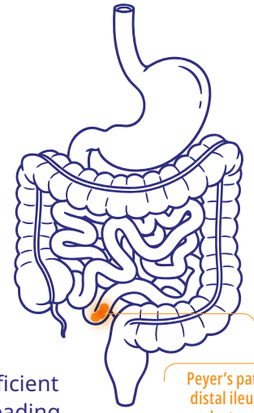
Adverse events should be reported. Reporting forms and information can be found at yellowcard.mhra.gov.uk or search for MHRA Yellow Card in the Google Play or Apple App Store. Adverse events should also be reported to Thornton and Ross Limited by emailing thorntonross@medinformation.co.uk or by calling 01484 848164.

STADA

IgA nephropathy: A major cause of CKD and kidney failure[4]

A disease of the kidney caused by pathogenic IgA production in the gut[5-7]

IgA nephropathy is characterised by the deposition of galactose-deficient IgA1 (gd-IgA1) antibody complexes in the glomerular mesangium, leading to irreversible glomerularsclerosis and loss of kidney function.[8]



Peyer's patches in the distal ileum produce galactose-deficient IgA1 antibodies[8]

50% of IgA nephropathy patients will reach kidney failure or death within 10-15 years[4]

UK RaDaR

Early monitoring and effective intervention is required to prevent unnecessary and irreversible nephron loss[8]

The 4-hit theory: prevailing theory on the development of IgA nephropathy[5-7]

1. Over production of mucosal galactose-deficient IgA1 antibodies which enter systemic circulation
2. Production of autoantibodies in response to recognition of aberrant galactose-deficient IgA1 antibodies, forming immune complexes
3. Deposition of immune complexes in the glomerular mesangium
4. Inflammatory cascade release of pro-inflammatory cytokines and fibrogenic factors in mesangial cells, causing irreversible kidney damage and renal failure

KDIGO recommends disease modifying treatment alongside traditional RASi and SGLT2i[1]

The focus of management in most patients should be to **simultaneously**:

- Prevent or reduce IgA immune complex formation and immune complex-mediated glomerular injury.
- Manage the consequences of existing IgA nephropathy-induced nephron loss.

IgA nephropathy at risk of progressive kidney function loss

▼ In all patients these should be considered simultaneously ▼

Manage the IgA nephropathy-specific drivers for nephron loss	Manage the generic response to IgA nephropathy-induced nephron loss	Cardiovascular risk reduction
Stop synthesis of pathogenic forms of IgA and IgA-immune complex formation	Stop IgA/IgA-immune complex mediated kidney injury	Reduce glomerular hyperfiltration and the impact of proteinuria on the tubulo-interstitium	Blood pressure control
Kinpeygo®		Lifestyle modification	
		RASi or DEARA ± SGLT2i	

Adapted from KDIGO Guidelines. (2025).[1]

To prevent further loss of nephrons and kidney failure, both CKD and immunopathology of IgA nephropathy should be addressed[8]

KDIGO 2025 guidelines[1]

- A patient with IgA nephropathy is at risk of progressive loss of kidney function if they have **proteinuria ≥ 0.5 g/d** (or equivalent). Treatment should be started in all cases
- Treatment goal: To reduce the rate of loss of kidney function to **< 1 ml/min per year** for the rest of the patient's life, or **maintain urine protein excretion at < 0.5 g/d (or equivalent), ideally < 0.3 g/d (or equivalent)**

Scan the QR code to access the 2025 KDIGO Guidelines

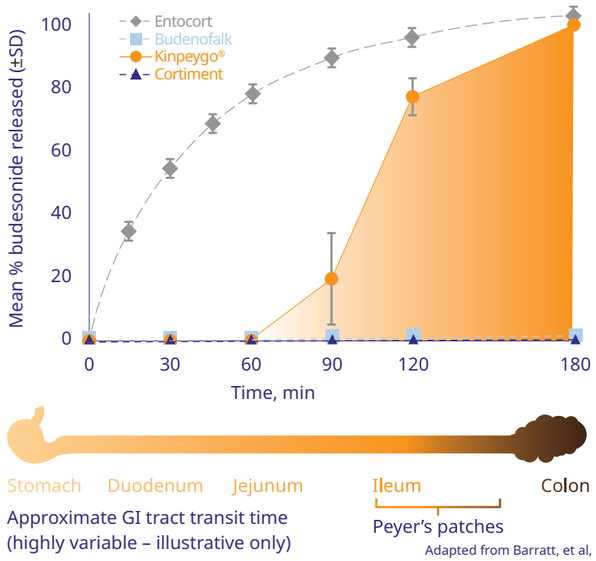
NICE guidelines update[2]

- Kinpeygo® an option to treat primary immunoglobulin A nephropathy (IgAN) in adults when they have a urine protein-to-creatinine ratio (UPCR) of 90 mg/mmol or more or a protein excretion of 1.0 g/day or more, and it is used as an add-on to optimised standard care.

The first and only disease-modifying treatment recommended by NICE as an option for use in primary IgA nephropathy in adults[2,3]

Kinpeygo® acts at hit 1: treating the underlying cause of IgA nephropathy, potentially delaying progression to dialysis or kidney transplant[2,12]

Unlike other budesonide formulations, Kinpeygo® targets the Peyer's patch-rich distal ileum to minimise disease progression and damage to kidneys[8,12]



Approximate GI tract transit time (highly variable – illustrative only)

Adapted from Barratt, et al, 2024.[8]

Designed to deliver budesonide topically in the ileum:[3]

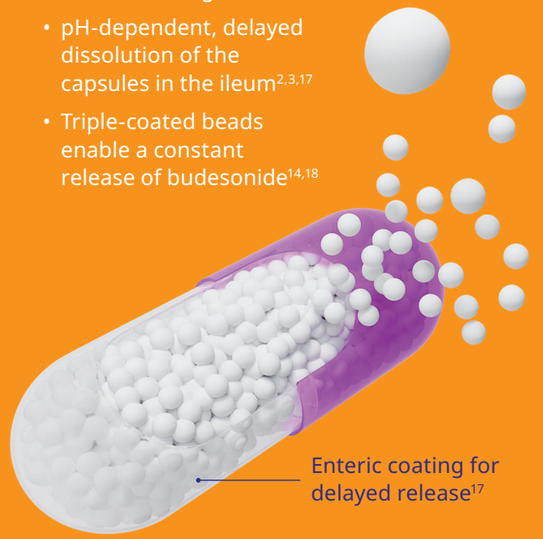
- Targeting the Peyer's patch-rich distal ileum to **minimise disease progression and damage to kidneys**[3,13]
- The formulation **reduces systemic circulation of gd-IgA**[2,15]
- Kinpeygo® has **90% first pass elimination**, which reduces the risk of AEs typically associated with the systemic administration of corticosteroids[3,16]

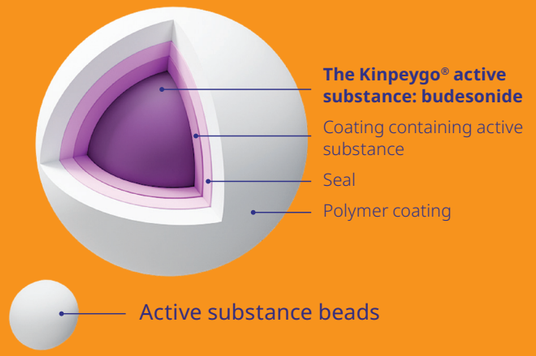
KINPEYGO® 4 mg
Modified-release hard capsules budesonide

Kinpeygo® acts with a targeted-release mechanism at the primary site of IgA1 production[12,13]

Kinpeygo®

- Hard capsule[1] with an enteric coating[17]
- pH-dependent, delayed dissolution of the capsules in the ileum[2,3,17]
- Triple-coated beads enable a constant release of budesonide[14,18]

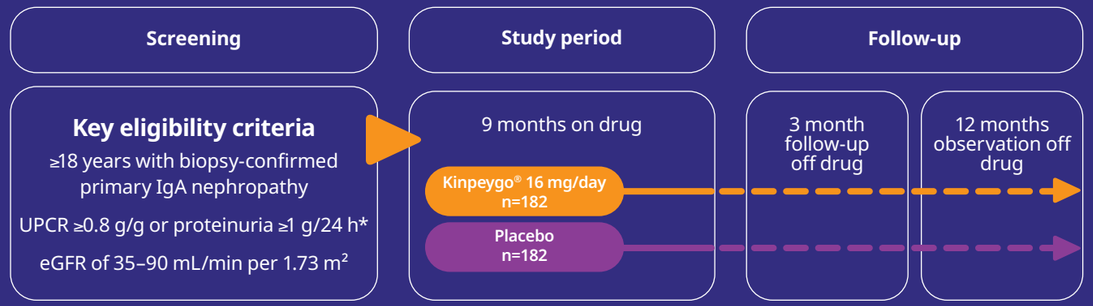




Posology and Administration[3]

1×	Once daily for 9 months
	16 mg/day (4 capsules)
	Capsules should be swallowed whole with water in the morning, at least 1 hour before a meal
	Patient should not double daily dose to make up for a missed dose
	28 capsule pack available to support dose tapering after 9 months of full treatment

The NefIgArd Phase III trial: 2-year results of Kinpeygo vs placebo in IgA nephropathy patients[13]



Patient demographics and baseline characteristics were balanced across the treatment groups and were representative of the intended primary IgA nephropathy population

Primary endpoint:

Time-weighted average of eGFR over 2 years

Total 2-year eGFR slope (supportive)

Secondary endpoint:

Time to confirmed 30% reduction in eGFR or confirmed kidney failure, 12- and 24-month UPCR and ratios and the proportion of patients without microhaematuria

Safety outcomes:

TEAEs, sAEs, vital signs, bodyweight, clinical laboratory variables, and physical examination findings

*In two consecutive measurements of the same parameter over ≥2 weeks.

Sustained, clinically meaningful impact in eGFR

- **Primary endpoint:** Kinpeygo® showed a +5.05 mL/min/1.73 m² time-weighted average eGFR benefit vs. placebo over 2 years (95% CI, 3.24–7.38, p<0.0001).
- After 2-years, Kinpeygo® reduced the decline in kidney function by ~50% vs placebo (-6.11 mL/min vs -12.00 mL/min).
- A 55% risk reduction in reaching a composite kidney endpoint (HR 0.45; 95% CI, 0.26–0.75, p=0.0014), exceeding thresholds predictive of long-term renal survival.

Robust and sustained reduction in proteinuria

- **Secondary endpoint:** UPCR change over 24 months. Kinpeygo® achieved a 40.3% reduction in UPCR from baseline over 2-years, vs 1.0% increase with placebo (p<0.0001).[19]
- A maximal ~50% reduction vs placebo was observed at 1-year confirming a strong and early therapeutic effect (95% CI, 41.6–56.6).
- The ~30% reduction in proteinuria seen at end of treatment (9 months) was maintained through 2-years.*

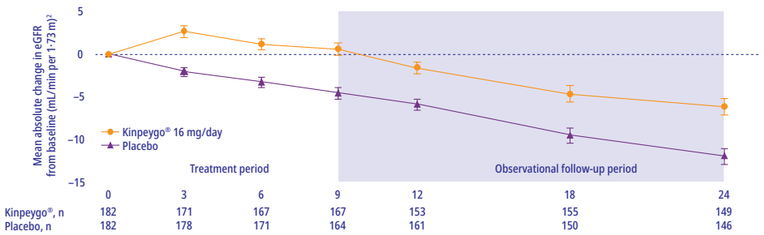


Figure 1: Mean absolute change in eGFR from baseline to 24 months (primary endpoint)

Adapted from Lafayette, et al, 2023.[13]

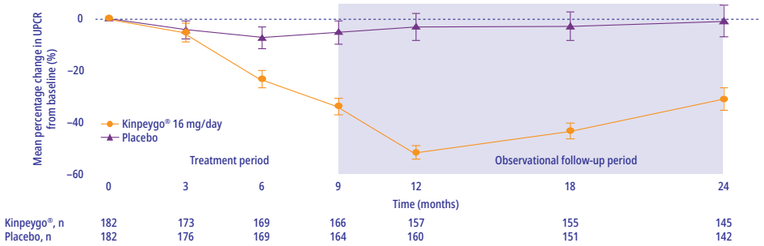


Figure 2: Mean percentage change in UPCR (g/g) from baseline to 24 months (secondary endpoint)

Adapted from Lafayette, et al, 2023.[13]

*Percentage change vs placebo in UPCR at 9 months: 30,0% (95% CI, 19.9–38.8) and at 24 months: 30.1% (95% CI, 16.4–41.5).

For additional resources or patient support materials, please scan the QR code or visit:
stadaspecialtybiosimilars.co.uk/kinpeygo

Kinpeygo® has a predictable and well-tolerated safety profile, as expected for a low-dose oral budesonide product[3,13]

- The most common TEAEs, such as peripheral oedema (17%), hypertension (12%), and acne (11%), were generally mild, non-serious, and resolved during or after treatment.
- 9% of patients discontinued due to AEs vs 2% with placebo; sAEs were rare and mostly unrelated to treatment, with no consistent pattern across organ systems.

Most commonly reported TEAEs* during treatment[13,19]

Adverse events	Placebo (n = 182)	Kinpeygo (n = 182)
Peripheral oedema	4%	17%
Hypertension	3%	12%
Muscle spasms	4%	12%
Acne	1%	11%
Headache	8%	10%
Nasopharyngitis	10%	9%
Face oedema	0.5%	8%

Adverse events	Placebo (n = 182)	Kinpeygo (n = 182)
Dyspepsia	2%	7%
Arthralgia	2%	7%
URTI	5%	5%
Insomnia	4%	5%
Fatigue	4%	5%
Rash	4%	5%
Increase in weight	3%	5%

Contraindications[3]

- Hypersensitivity to the active substance or to any of the excipients
- Patients with severe hepatic impairment (Child-Pugh Class C)

Treatment discontinuation[3]

- Dose should be reduced to 8 mg once daily for the last 2 weeks of therapy
- Dose may be reduced to 4 mg once daily for an additional 2 weeks, at the discretion of the treating physician
- Re-treatment may be considered at the discretion of the treating physician†

AE, adverse event; CI, confidence interval; CKD, chronic kidney disease; DEARA, dual endothelin angiotensin receptor antagonist; eGFR, estimated glomerular filtration rate; gd, galactose-deficient; GI, gastrointestinal; HR, hazard ratio; IgA, immunoglobulin A; KDIGO, Kidney Disease: Improving Global Outcomes; RaDaR, National Registry of Rare Kidney Diseases; RASi, renin-angiotensin system inhibitor; sAE, serious adverse event; SD, standard deviation; SGLT2i, sodium-glucose transport protein-2 inhibitor; TEAE, treatment-emergent adverse event; UPCR, urine protein-creatinine ratio; URTI, upper respiratory tract infection.

*Summary of TEAEs during treatment period (≥5% in the Kinpeygo arm); †Safety and efficacy of treatment with subsequent courses of Kinpeygo® have not been established.

1. KDIGO. Clinical Practice Guideline for the Management of IgA Nephropathy (IgAN) and IgA Vasculitis (IgAV). Available at: https://kdigo.org/guidelines/iga-nephropathy/. Last accessed: February 2026; **2.** NICE. Technology appraisal guidance TA1128: Targeted-release budesonide for treating primary IgA nephropathy. Available at: https://www.nice.org.uk/guidance/ta1128. Last accessed: February 2026; **3.** Kinpeygo® SmPC. Available at: www.medicines.org.uk/emc/product/15393/smpc. Last accessed: February 2026; **4.** Pitcher D, et al. Clin J Am Soc Nephrol. 2023;18(6):727–38; **5.** Barratt J, et al. Front Med (Lausanne). 2024;11:1461879; **6.** Zhang Y-M, Zhang H. Clin J Am Soc Nephrol. 2018;13(10):1584–6; **7.** Suzuki H, et al. J Am Soc Nephrol. 2011;22(10):1795-803; **8.** Barratt J, et al. Drug Des Devel Ther. 2024;18:3415–28; **9.** Kidney Care UK. IgA nephropathy. Available at: https://kidneycareuk.org/kidney-disease-information/kidney-conditions/iga-nephropathy/. Last accessed: February 2026; **10.** Zaidi O, et al. BMC Nephrol. 2024;25(1):136; **11.** Rout P, et al. In: StatPearls [Internet]: IgA Nephropathy (Berger Disease) Treasure Island (FL): StatPearls Publishing; 2025; **12.** Fellström BC, et al. Lancet. 2017; 389(10084):2117-27; **13.** Lafayette R, et al. Lancet. 2023;402(10405):859-70; **14.** Watts P, Smith A. Expert Opin Drug Deliv. 2005;2(1):159-67; **15.** Edsbacker S. et al. Gastroenterology. 1993;104(4 Suppl): A695; **16.** Wang J, et al. Kidney Int Rep. 2024;9(9):2705–17; **17.** Tarpeyo US Prescribing Information. Calliditas. 2023; **18.** Tarpeyo HCP. How Tarpeyo Works. Available at: https://www.tarpeyohcp.com/mechanism#designed-with-targeted. Last accessed: February 2026; **19.** Lafayette R, et al. Lancet. 2023;402(10405):859-70. Suppl. Appendix.

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